Cardiac Arrest During Anesthesia

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SUMMARY

In cases of acute cardiac arrest from transient or reversible causes resuscitation is a distinct possibility. Prompt action is of the utmost importance.

The sequelae of cardiac arrest will depend on the degree of anoxia that has developed and the amount of irreversible damage to brain tissue. Efficient manual artificial circulation, begun immediately, can prevent such damage.

Four cases of cardiac arrest are described. Sudden circulatory collapse, which may or may not imply cardiac arrest, is not uncommon during surgical procedures. Usually the patient recovers quickly when treatment is prompt.

WHEN does death occur? There is no immediate criterion indicative of the transition from life to death. Years ago, cessation of respiration was considered synonymous with death. It was a common practice to hold a mirror in front of a patient's face and if there was no condensation of moisture from exhaled air, he was pronounced dead. When it was realized that the artificial maintenance of respiration could prevent death, the fallacy of this view became apparent. Cessation of cardiac activity superseded cessation of respiration as the criterion of death. But again a fallacy became apparent. Some patients were revived after cardiac arrest. Jut as a community is not dead when its transportation system fails, so the body is not dead when respiration or circulation fail. But just as a community will disintegrate and cease to be a community when the resulting starvation ravages its population and disrupts all coordinated effort, so it is with the body when the effects of respiratory or circulatory failure result in irreparable damage. Death does *not* occur until after there has been so much irreversible damage to nervous tissue that restoration of coordinated vital functions is impossible.

Respiratory arrest and cardiac arrest are very similar functional disorders and the principles applicable to one are equally applicable to the other. The apparent differences are chiefly those of relative urgency and relative accessibility. Depending on circumstances, respiratory arrest may persist from perhaps two to twelve minutes before irreversible damage results, because the oxygen in the lungs and blood acts as a reservoir. On the other hand, cardiac arrest will probably result in some irreversible damage in from two to four minutes. To counteract respiratory arrest by artificial respiration requires only moments because the respiratory mechanism is accessible. To counteract circulatory arrest by manual circulation is likely to require minutes. The margin of safety in this condition is much narrower.

It is fairly obvious that if the heart fails because of irreversible contributing circumstances such as severe hemorrhage, shock, or embolus, efforts at cardiac resuscitation are well-nigh hopeless. On the other hand, in cases of acute cardiac arrest from transient or reversible causes, resuscitation is a distinct possibility and any restorative measure, no matter how drastic, is justifiable.

As stated by Thompson and co-workers,[8] "acute cardiac cessation is conveniently classified into two groups: (1) arrest of stimulus formation or the so-called pace-maker failure, the result of which is cardiac standstill and (2) electrodynamic dissolution of the cardiac cycle, of which ventricular fibrillation is an example." The chances of successful resuscitation are greater in the case of cardiac standstill than in the case of ventricular fibrillation. Fortunately most cases fall into the former group. It is impossible to differentiate the two clinically unless the heart is already exposed or an electrocardiogram is in process.

ETIOLOGY

There are various circumstances which may lead to acute cardiac arrest:

1. *Reflex arrest* due to vagovagal stimulation. Such stimuli may arise from surgical trauma in the region of the aorta, the hilus of the lung, the carotid sinus or the vagus nerves. Weeks and co-workers[10] reported a case of cardiac arrest immediately following section of the vagus. It may also result from traction reflexes. Similarly, it has been postulated that anesthetic procedures irritating the tracheobronchial tree, such as intubation, may on occasion excite such reflexes. In certain cases, patients may be put into peculiar positions which result in reflex cardiac arrest.

2. *Direct trauma* to the heart, such as may occur during operations on the pericardium or heart or may be caused by inadvertent pressure of retractors on the heart or aorta, may cause cardiac arrest.

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3. *Overdosage of anesthetic agents* may depress the circulation, either by direct effect on the heart or by vasodilation. Chloroform, ethyl chloride and cyclopropane may have direct effects on the heart, particularly by altering the conduction mechanism or, in the case of chloroform and ethyl chloride, by direct depression of the myocardium.

4. *Ventricular fibrillation.* The heart, sensitized by certain anesthetic agents, may react by ventricular fibrillation if subjected to an overdose of epinephrine. This epinephrine may be introduced by injection or may be endogenous, coming from the adrenal glands if there is stimulation or excitement during light anesthesia.

SIGNS OF CARDIAC ARREST

It is sometimes difficult to be certain whether the heart has actually stopped during a surgical procedure or whether its beats are so feeble that there is little evidence of circulation. Therefore all possible means should be utilized to make a rapid evaluation of the situation. The following points should be observed:

1. *Absence of pulsation.* If the anesthetist is feeling the pulse at the time cardiac arrest occurs, he will notice the sudden absence of pulsation. More often, however, other indications precede this and call the anesthetist's attention to the emergency. He then attempts to feel the pulse but finds that he cannot detect it. There will also be no visible pulsation in the neck. The surgeon may have access to a large artery which he may palpate to determine if there is any pulsation.

2. *Absence of blood pressure.* If attempts are made to determine the blood pressure, it will be found that no audible or visual pulsations occur.

3. *Respiratory arrest.* The most common initial indication of cardiac arrest is the almost immediate cessation of respiration due to cerebral anoxia. There may be a few gasps preceding this. In all cases of respiratory arrest, one of the anesthetist's first obligations is to feel for the pulse and determine if the circulation is satisfactory.

4. *Pallor or cyanosis* quickly follows cardiac arrest. The actual color will depend somewhat on the peripheral distribution of blood, and the position of the patient is a factor. The blood will gravitate to the dependent parts of the body. If the head is elevated, pallor of the upper part of the body may be expected. If the head is lowered, congestion and deep cyanosis may develop.

5. *Cardiac sounds* cannot be heard over the precordium.

6. *Absence of bleeding* in the wound may be observed. The surgeon may be requested to incise a moderate-sized artery to see if it will bleed.

7. *Direct observation of heart.* If the surgeon is performing a transthoracic operation, he may observe the state of the heart directly. If he is performing an abdominal operation, he may palpate the heart from below the diaphragm.

8. *Dilation of the pupils* may develop as anoxia progresses.

9. *The capillary refill time* as determined by pressure on the skin, and the duration of the resulting pallor, may be of some significance.

PROPHYLAXIS

Since in many cases cardiac arrest results from vagal stimulation, it is advocated that adequate atropinization be used when there is serious likelihood of stimulation in one of the more sensitive areas. Similarly, blocking of the afferent stimuli by infiltration with a local anesthetic agent in areas such as the hilus of the lung may prevent the condition. In cases in which trauma near the hilus of the lung, the heart or the pericardium causes arrhythmia, the injection of 5 cc. to 10 cc. of 1 per cent procaine solution intravenously may relieve the condition and prevent cardiac arrest. Direct application of procaine to the heart during cardiac operations is sometimes utilized, although the value of this is more questionable.

TREATMENT OF ACUTE CARDIAC ARREST

As has been emphasized by Bailey,[2] time is of the utmost importance and a preconceived plan of action should be followed in order to avoid any unnecessary delay. Bailey stressed the value of keeping an accurate check on the time which has elapsed from the onset of the emergency, and he advocated that one person in the operating room be delegated at once to be the time-keeper and to call off the passing minutes. Whether this is done or not, it is vitally important that the duration of the emergency be carefully observed and that the routine of action be gauged according to the time which has elapsed. All anesthetists should have some such routine in mind, and it will probably be found that surgeons will respond and cooperate if the anesthetist demonstrates his knowledge of the situation and his competence to direct procedures. The following is an outline of a routine which may be followed.

1. *Immediate.* As soon as cardiac arrest is suspected, several things should be done almost simultaneously. Since only a matter of seconds need elapse until all are instituted, the exact order is unimportant:

(a) Notify the surgeon of the suspected emergency.

(b) Lower the head of the table to a Trendelenburg position of 10 to 15 degrees.

(c) Discontinue the administration of the anesthetic agent and substitute pure oxygen or air.

(d) Correct any situation which has possibly contributed to the emergency. The possibility that an anesthetic agent is being administered inadvertently, as by leaving the valve of an ether bottle open, should be considered. Patients have died because anesthetists have persisted in resuscitative efforts without realizing that they were continuing to administer a hgh concentration of ether or that they were erroneously using nitrous oxide instead of

oxygen. Any recent manipulation by the surgeon which might have precipitated a vagovagal reflex should be discontinued. Any change of position which has been followed by circulatory collapse or cardiac arrest should be corrected.

(e) The anesthetist should immediately look at his watch and make an accurate note of the time of onset of the emergency. An alternative is to follow Bailey's method and delegate someone in the room to watch the time.

(f) Most important of all, artificial respiration should be instituted at once and continued throughout, with two exceptions noted below. If the anesthetist is sure that there is no error in the apparatus, inflation of the lungs with pure oxygen by means of pressure on the breathing bag is a desirable method. If he is in doubt about the machine or does not have one immediately available, then mouth-to-mouth or mouth-to-nose respiration is the best substitute. If the patient is anesthetized with an agent which is eliminated through the lungs, provision must be made for a free elimination of the exhaled atmosphere. Thus, in case of overdosage of an inhalation agent, one must not use the carbon dioxide absorption technique with only a maintenance flow of oxygen. The technique may be used but there must be a sufficiently rapid flow of oxygen (at least six liters per minute) to provide rapid elimination of the exhaled atmosphere. It was observed in animal experiments by Thompson and co-workers[8] that artificial respiration with alternating positive and negative pressures, such as is produced by certain resuscitators, is more effective in restoring cardiac activity than mere intermittent inflation. However, such resuscitators may not be available. Artificial respiration by means of positive pressure has a distinct effect in producing some peripheral circulation. This is brought about by the increased intrathoracic pressure compressing the heart and squeezing the blood out. Although in some cases cardiac arrest may be relieved merely by this form of cardiac stimulation, it must not be assumed that vital functions are maintained by this propulsive effect on the blood. Volpitto and co-workers[9] demonstrated that the two most important parts of the circulation—namely, that in the cerebral and in the coronary arteries—are not influenced by this kind of artificial circulation.

If possible, an endotracheal tube should be inserted. However, delay for this procedure cannot be countenanced unless inflation of the lungs is otherwise impossible because of obstruction. The endotracheal tube has several advantages. It insures a free respiratory exchange and it obviates the possibility that the stomach may be inflated with oxygen which passes down the esophagus.

2. *During the first minute.* If positive evidence of cardiac arrest is not immediately available, the first minute may be occupied with efforts to determine the actual situation. If the surgeon can immediately feel or see the heart and verify its inactivity, the procedure described under (4) below should be started immediately. Occasionally an arrested heart may be stimulated by thumping over the precordium. Therefore this type of stimulation may be attempted during the first minute but it should not in any way supersede the other efforts described.

3. *During the second minute.* If evidence of cardiac activity has not been elicited, it should be assumed that there is cardiac arrest and this minute should be utilized to make preparation for gaining direct access to the heart, either through an upper abdominal or thoracic incision. The surgeon should be notified of the necessity for this and should make his preparations. If possible, the field should be disinfected and sterile drapes and instruments obtained. However, since nothing can take precedence over the problem of reestablishing circulation, these details may be modified according to facilities available. During this minute, further observations regarding pulse, heart sounds, etc., are made so that if cardiac activity resumes it will be noted.

4. *During the third minute.* If, at the end of two minutes, there is still no evidence of circulation, the surgeon should proceed with the incision which he has elected and gain access to the heart. If, under direct observation or palpation, no heart beat is apparent, manual artificial circulation should be instituted and maintained until spontaneous contractions return or all efforts are considered to be of no avail. The rhythmical squeezing of the heart has two purposes: first, to propel blood and maintain circulation and, second, to stimulate the cardiac muscle in the hope of restoring spontaneous activity.

When artificial respiration and manual artificial circulation are maintained, the urgency of the situation is relieved. Oxygenation of vital tissues is maintained. Ample time may be taken to relieve the cardiac arrest by other means if necessary. Adams and co-workers[1] reported successful resuscitation with complete recovery after 20 minutes of cardiac arrest. It must be emphasized that in that case the heart was under direct observation and manual artificial circulation was instituted immediately after the arrest occurred so that oxygenation of the tissues was not impaired throughout the 20 minutes. (It is well to note in passing that the term "cardiac massage," which so often is used to describe the manual rhythmical squeezing procedure, is misleading. In one case, to be described later, an orthopedic resident heroically responded to the anesthetist's request that an abdominal incision be made and "cardiac massage" performed. However, it appears that the manipulation he carried out was indeed "massage" in the generally understood meaning of the word.)

The surgical approaches to the heart are (1) transperitoneal, either subdiaphragmatic or transdiaphragmatic; and (2) transthoracic, either extrapleural or intrapleural.

For the subdiaphragmatic approach, a high midline or left paramedian incision should be made. The surgeon then inserts one hand under the diaphragm and places the other over the precordium. The heart is then squeezed between the hands forc-

ibly and rhythmically at the rate of about 60 times per minute. As this method is tiring and not completely efficient, the transdiaphragmatic approach may be added: The diaphragm and adjacent pericardium are incised to permit one hand to be inserted. The heart is grasped in it and rhythmic squeezing continued.

The transthoracic approach is regarded as the one of choice by Barber and Madden.[3] Naturally, if an intrathoracic operation is in progress, this provides a transpleural approach, and access to the heart is obtainable immediately. If it is necessary to incise the thorax, the following method described by Barber and Madden is simple and satisfactory: "Exposure of the heart through a transverse incision in the third or fourth interspace is adequate and readily performed. The incision extends from the anterior surface or left border of the sternum, transversely to the left nipple or anterior axillary line. The incision is deepened through the underlying fascia and muscle layers and the corresponding intercostal space. The adjacent costal cartilages above and below are sectioned and the corresponding ribs widely retracted. The production of pneumothorax is avoided if possible, but it is of no undue consequence if it should occur."

This approach has several advantages. It permits direct observation of the heart so that its actual condition can be verified, and direct application or injection of drugs is facilitated. The maintenance of manual artificial circulation is easier because of the more adequate access to the heart.

5. *Ventricular fibrillation.* If ventricular fibrillation is apparent on inspection of the heart or if it is seriously suspected, certain specific measures aimed at stopping the fibrillation may be instituted. One is to inject 5 to 10 cc. of 1 per cent procaine solution into the right auricle or the left ventricle. This reduces the irritability of the myocardium and facilitates response to normal pace-maker activity. Another method is the use of electrical stimulation of the heart, as done in animal experiments by Prevost and Battelli[7] and as described for application to humans by Beck.[5] Two large electrodes are placed on opposite sides of the heart and shocks of 1 to 1.5 amperes produced. The shock is applied for a fraction of a second. If one shock does not stop ventricular fibrillation, a second and third may be given.

6. *Analeptic drugs.* With artificial respiration and manual artificial circulation in progress, there is no urgency about using other and perhaps hazardous methods. However, intracardiac injection of an analeptic drug may be resorted to if other measures appear to have no effect. The mere passage of a needle through the myocardium may be sufficient to stimulate normal cardiac activity. The injection should be made into the right auricle, since this brings the stimulus near to the pace-maker. A 4½-inch needle should be inserted through the second or third intercostal space immediately to the right of the sternum and directed posteriorly and inferiorly until blood can be withdrawn from the cavity of the heart. The injection is then made. Some investigators have suggested the injection of some of the analeptic drug into the myocardium. However, if epinephrine is used, there is some danger that it may result in local ischemia and infarction. Trauma to a coronary artery caused by the needle, although unlikely, can occur. Sequelae of such a complication should be kept in mind postoperatively. Analeptic drugs suggested have included epinephrine, Coramine® and Metrazol.® There is a tendency to give too much; 0.5 cc. of epinephrine in 1:1,000 solution should be sufficient. If there is no response in a reasonable time, the injection may be repeated. In many cases the injection of epinephrine into the heart has been used before resorting to manual artificial circulation, and in some instances it has proved effective. However, there are reasons why this is injudicious and the apparently more radical method of gaining direct access to the heart should have priority. In the time taken to obtain and to inject epinephrine and in the further delay to see if it has been effective, anoxemia may progress to the point of causing irreversible damage before manual artificial circulation is eventually instituted. Also, epinephrine may cause ventricular fibrillation. Thus if it is injected into a heart which is still beating feebly or which is in standstill, it may cause ventricular fibrillation from which there is much less likelihood of recovery.

The addition of even low concentrations of carbon dioxide to the inhaled atmosphere has been observed by Eastman and Kreiselman[6] to be detrimental.

7. *Interruption of artificial respiration.* As previously mentioned, there are two occasions during which the artificial respiration, otherwise maintained continuously, should be interrupted. One is during the incision of the chest wall or abdominal wall. If the chest wall is being incised, inflation of the lung may result in inadvertent incision of the pleura and lung. During incision of the abdominal wall, respiratory movements may increase the possibility of accidental incision of the stomach. Incidentally, if the lungs have been inflated during the procedure without an endotracheal tube being in place, there is every likelihood that the stomach will be distended and it is advisable to warn the surgeon of this fact. The second occasion on which artificial respiration should be interrupted is during the insertion of a needle into the heart. Inflation of the lung at the wrong time may result in laceration of the pleura or puncture of the lung.

8. *Inflation of the stomach.* As already mentioned, the stomach is likely to be inflated during some kinds of artificial respiration. It may be deflated from time to time during the resuscitative procedure by pressure on the epigastrium. At the end of the resuscitative efforts, whether the patient has recovered or been pronounced dead, the stomach should be deflated if it appears to be distended. This may require the insertion of a gastric tube. If the patient

is alive, the distended stomach may hamper respiration and circulation. If the patient is dead, the pathologist performing the autopsy may be misled into a diagnosis of acute dilation of the stomach if it has not been deflated.

9. *Duration of resuscitative efforts.* If there was no undue delay in instituting artificial circulation and if there is no added cause of anoxia, the resuscitative efforts may be continued for at least an hour before hope is abandoned.

SEQUELAE OF PROLONGED CARDIAC ARREST

Cardiac arrest is not in itself the cause of death or of subsequent pathologic conditions. It is only a functional disturbance. It is the resulting anoxia that causes damage, particularly to the nervous system where the damage rapidly reaches an irreversible degree. If this damage is sufficiently extensive, resuscitative measures will fail completely. Unfortunately, various degrees of damage resulting in permanent neurologic changes may occur. In many cases, cardiac and respiratory activity may be restored temporarily only to fail hours or days later. Hyperpyrexia may develop. In many other cases the patient may live for an indefinite period but with neurologic damage as manifest by dementia, spastic paraplegia, blindness, etc. Weinberger and co-workers,[11] working on cats, clamped the pulmonary artery for periods ranging from two minutes to ten minutes and fifteen seconds and analyzed the amount of damage resulting. In these experiments, arrest of the circulation for three minutes and ten seconds or less resulted in no neurologic disturbances. When the period was extended to three minutes and 25 seconds, changes in behavior and psychic functions occurred. After six minutes of circulatory arrest, vision and sensation were permanently impaired. After seven minutes and 35 seconds, there were permanent dementia, blindness, sensory and auditory defects, motor and postural defects and reflex abnormalities. After eight minutes and 45 seconds or longer, life could not be restored for more than a few hours. The human brain is probably more susceptible to anoxia than is that of cats.

In cases in which death occurs within a few hours after restoration of cardiac activity, there is usually pulmonary damage resulting in pulmonary edema.

INCIDENCE

It is impossible to estimate the actual incidence of cardiac arrest during anesthesia. There are many cases in which absence of pulse and blood pressure and respiration and other signs point to this diagnosis but in which the patient recovers promptly during the first minute or so of treatment. Barber and Madden[4] stated in 1945 that they had been able to collect from the literature reports of a total of 143 cases of "cardiac massage," with complete recovery in 48 cases (33 per cent). Many more cases must have occurred which are not in the literature.

At the Research and Educational Hospitals from September 1937 until May 1947, there were nine cases in which manual artificial circulation was carried out following sudden cessation of cardiac activity. One of the patients had a large pulmonary embolus and could not have recovered. Of the eight remaining, two made complete recoveries and two recovered temporarily but died, one in two hours and the other in 69 hours. During the same period there were eight other cases in which the patient died but in which radical measures were not tried. In several of those cases death was caused by neurologic disturbance.

The following cases are illustrative of certain features previously described.

A three-year-old boy who had been born with amputation of both thighs and was scheduled for reamputation of one thigh was anesthetized by open drop technique with ethyl chloride and ether. The ethyl chloride was discontinued early in the induction. A tourniquet was applied to the thigh prior to the operation. Thirty minutes after the operation started, the child's condition appeared to be good. The tourniquet was removed and the patient immediately became pale and pulseless and did not breathe. The anesthetist did not institute artificial respiration or other resuscitative measures and two or more minutes elapsed before another anesthetist who was called in lowered the patient's head and instituted mouth-to-mouth breathing. When there was no improvement, the orthopedic resident was asked to perform "cardiac massage." An abdominal incision was made and the cardiac massage was begun subdiaphragmatically. (In this case it appeared that the surgeon rubbed the heart in the manner of a masseur and did not institute efficient manual artificial circulation.) A few cardiac beats occurred but the action did not persist. Epinephrine, 0.5 cc., was injected into the right auricle. Spontaneous cardiac activity was restored, and spontaneous respirations followed. At first they were gasping but gradually they improved. Probably at least ten minutes had elapsed from the time of cardiac arrest until efficient circulation was restored. The abdomen was closed and the orthopedic operation completed. The patient was returned to bed and put in an oxygen tent. Twitching of the eyes, face and neck and hands was observed and pulmonary edema developed. The patient died two hours after the end of the operation. At autopsy, atelectasis of the right lung and congestion of the left, secondary to the pulmonary edema, were noted. A small incision in the stomach which had not been evident to the surgeon was observed. This had no bearing on the patient's death.

This case illustrates the importance of instituting effective treatment early. If artificial respiration had been started immediately, and efficient manual artificial circulation instituted within three minutes, the cerebral and pulmonary damage due to anoxia would probably not have occurred and restoration of cardiac activity might have restored the child to a healthy condition. The misleading implication of the term "cardiac massage" is illustrated, as is the importance of care not to damage the stomach in such procedures.

The patient was a 51-year-old obese female with cholecystitis and obstructive jaundice. There was no record of cardiac disturbance. Before anesthesia the blood pressure was 120 mm. of mercury systolic and 80 mm. diastolic. The pulse rate was 84. Pontocaine, 16 mg., was given intraspinally and anesthesia reached the fourth thoracic seg-

ment. Neosynephrine, 0.5 cc., then was given. Before the start of the operation, the blood pressure was 90 mm. of mercury systolic and 50 diastolic. Ten minutes after the beginning of the operation, just after the gallbladder lift had been raised and while the surgeon was palpating above the liver, the blood pressure could not be obtained, the pulse could not be felt and respirations ceased. Artificial respiration was started immediately. The surgeon palpated the heart and could detect no activity. Manual artificial circulation was instituted immediately by the subdiaphragmatic route and cardiac action returned very quickly. Following this, 1 cc. of epinephrine in 1:1,000 solution was injected into the heart. Spontaneous respirations were resumed. The whole episode probably lasted about two minutes. Following it the blood pressure rose to 200 mm. of mercury systolic and 140 mm. diastolic, then in the course of 20 minutes gradually decreased to 80 mm. systolic and 50 mm. diastolic. The stomach, which had been inflated during the artificial respiration, was deflated by insertion of a gastric tube. The remainder of the operation was uneventful. At the end of the operation the patient was awake and responsive and there was no evidence of damage. Oxygen therapy was given and recovery was satisfactory, although it was complicated by some consolidation of the upper lobe of the right lung.

This case illustrates the desirable result which may be obtained when artificial circulation is instituted promptly. The fact that an abdominal operation was in progress contributed to the speed with which this treatment was started. The injection of epinephrine was unnecessary and therefore injudicious. The cause of the arrest was probably reflex due to the surgical manipulation above the liver. That it occurred during spinal anesthesia is of interest.

A 17-year-old girl with mediastinal tumor was otherwise in good physical condition. The preoperative blood pressure was 120 mm. of mercury systolic and 55 mm. diastolic, and the pulse rate was 88. The patient was anesthetized with cyclopropane and intubated with an orotracheal tube with a cuff. Anesthesia was maintained in the low second plane. Controlled respirations were started 12 minutes after the operation started. The left pleural cavity was opened and the surgeon proceeded with the dissection of the mediastinal tumor. A blood transfusion was started 50 minutes after the beginning of the operation. An hour and 15 minutes after the start of the operation, while the surgeon was working in the left hilar region, the temporal pulse suddenly disappeared and the blood pressure could not be obtained. About 30 seconds later the surgeon was asked to examine the heart which was hidden by packs and a retractor. It was found to be in a state of standstill. Manual artificial circulation was immediately started, artificial respiration having been carried on during the entire time. About two minutes later, spontaneous cardiac activity was resumed, the heart rate being 104. Controlled respirations were continued and the operation completed 90 minutes later. Throughout this period, the pulse and blood pressure could not be obtained but the color of the blood was good and cardiac action could be observed. No addition of the anesthetic agent was necessary. There was some difficulty in administering fluids rapidly. The pulse and blood pressure were not obtainable until an hour after completion of the operation. At that time the pulse rate was 150 and the systolic blood pressure 80 mm. of mercury. The pulse rate rose as high as 200 but later returned to 130. The blood pressure slowly rose to 90 mm. of mercury systolic and 70 mm. diastolic. Postoperatively the temperature ranged from 101° to 104° F. On the third day tension pneumothorax was noted and several hundred cubic centimeters of fluid and air were removed. There were no signs of neurologic damage and the patient left the hospital in good condition.

In this case the cardiac arrest was probably due to reflex stimulation in the region of the hilus of the lung. The local use of procaine in this area or the intravenous injection of procaine or the administration of a large dose of atropine might have prevented the cardiac arrest. Prompt initiation of the artificial circulation resulted in complete recovery. When cardiac arrest is suspected, the anesthetist should not hesitate to interrupt the surgeon and call for his cooperation in making the diagnosis and in instituting treatment.

A 59-year-old woman with left hemiparesis as a result of a previous cerebral accident was operated upon for repair of evisceration four days after colostomy. Anesthesia was induced with cyclopropane with the canister out. Three minutes or less after the beginning of anesthesia the respirations became inadequate, the color was poor, and the pulse feeble. The anesthetic agent was stopped and pure oxygen was administered. A minute later neither blood pressure nor pulse was obtainable and respirations ceased. An endotracheal tube was inserted and artificial respiration was instituted immediately. The surgeon was notified of the seriousness of the situation and after three minutes of repeated urging he palpated the heart from below the diaphragm and reported no pulsation. Manual artificial circulation was then instituted. In 20 seconds a few weak beats were felt and five minutes later the heart was beating regularly at 120 beats per minute. Neosynephrine, 3 mg., was then given intramuscularly. Spontaneous respiration began 10 minutes after resumption of spontaneous cardiac activity. Oxygen was administered while the wound was being closed. When the patient left the operating room the blood pressure was 80 mm. of mercury systolic and 40 mm. diastolic, and the pulse rate was 120. The endotracheal tube was left in place and oxygen was continued on the ward. The patient died 69 hours postoperatively without having regained consciousness.

The previously existing brain damage probably made the nervous tissue more susceptible to anoxia, thus accounting for the fatal outcome in spite of the relatively early institution of manual artificial circulation. This case illustrates well the need for complete cooperation among the members of the operating room team and for a course of action based on a plan previously discussed.

CIRCULATORY COLLAPSE

There are many cases in which sudden circulatory collapse becomes apparent. It frequently occurs during rapid induction of general anesthesia. If neglected, it can go on to a more serious or even fatal outcome, whereas prompt initiation of artificial respiration and lowering the head results in prompt recovery. Whether any cases of circulatory collapse can truly be classified as cardiac arrest is uncertain, but undoubtedly many would rapidly fall into that classification if treatment were delayed.

A six-month-old girl was scheduled for lens extraction. Anesthesia was started with ethyl chloride by the open drop technique. Probably the ethyl chloride was allowed to flow

too freely and too long. About the time that the patient was in low second stage, ether was started but a little more ethyl chloride was added with it. Excess mucus developed. Obstruction, with little effort to breathe, followed. Respiration ceased, the pulse was not palpable, the heart beat was neither palpable nor audible, and there was pronounced prolongation of the capillary refill time. To all appearances the heart was in complete arrest. A few efforts to squeeze the chest and produce artificial respiration were ineffective. The child was turned for a brief period to the prone position to drain mucus from the mouth. An airway was inserted and mouth-to-mouth repiration was started. Spontaneous respirations were resumed after a few inflations and evidences of adequate circulation returned almost immediately. During the mouth-to-mouth respiration it was noted that considerable air was being blown into the stomach. This was expelled once by pressure on the epigastrium but the stomach became distended again; and after the child resumed normal respirations, the air could not be expelled in the same way. It was released through a Levine tube passed into the stomach. Anesthesia was continued for some time with ether, and was uneventful. The operation was cancelled not because of the circulatory collapse but because the surgical preparation had not been adequate. The child was anesthetized on a subsequent occasion without untoward effect.

In this case the circulatory collapse was probably due to some direct depressant effect of ethyl chloride on the myocardium. In such cases it appears that further anesthesia with another agent and continuance of the operation is not contraindicated because the episode does not necessarily indicate any inherent circulatory weakness.

An obese 61-year-old male patient was to have gastric resection for carcinoma. Edema of the ankles and pulmonary emphysema were noted preoperatively. The blood pressure was 130 mm. of mercury systolic and 80 mm. diastolic. Anesthesia was begun with cyclopropane by the closed technique. Orotracheal intubation was done with some difficulty, after which a canister was inserted. A few minutes after intubation the patient's pulse and blood pressure were unobtainable and respirations ceased. The lungs were inflated several times by pressure on the bag and almost immediately spontaneous respirations were resumed, the pulse became palpable at a rate of 70 and the blood pressure was 120 mm. of mercury systolic and 80 diastolic. At this time ether was started and cyclopropane gradually eliminated. The blood pressure rose to 160 mm. systolic and 90 diastolic, and the pulse rate to 108. Throughout the next three hours there was a gradual fall in blood pressure to 65 mm. systolic and 50 diastolic and a gradual slowing of the pulse to 56. No anesthetic agent was added for the last hour of anesthesia, and during the four and one-half hours of the operation the patient received 800 cc. of saline solution and 1,400 cc. of blood. Just as the last suture was being put in, the anesthesia was discontinued and a catheter was inserted through the endotracheal tube to aspirate secretions. The patient became cyanotic and respirations ceased. Artificial respiration with oxygen was started. The blood pressure and pulse could not be obtained. No radical procedures were instituted; and when the circulation did not return, the patient was pronounced dead.

At autopsy, extreme fatty infiltration of the heart was noted. The auricles particularly were almost replaced by fatty tissue. There was pronounced atherosclerosis of the coronary arteries. Partial atelectasis of the left lung was present; it was probably a terminal development, unrelated to the circulatory failure.

This case illustrates several interesting points. The anesthetists did not regard the initial circulatory collapse as an ominous sign and did not call it to the attention of the surgeon, who would have been willing to cancel or curtail the operative procedure. As the patient was obese, fatty infiltration of the myocardium might have been suspected and the emphysema might have been considered as a contraindication for anesthesia and operation. In such a case the early circulatory collapse should have been taken as a warning that the patient could not tolerate much trauma.

Circulatory collapse is a dangerous complication and may lead directly to death. Once the acute episode has been relieved, evaluation of its significance depends on various circumstances. If the patient is young and healthy and the episode has occurred during induction with an agent which may directly depress the heart, such as ethyl chloride, it may be assumed that it does not signify serious circulatory disease, and it is usually justifiable to proceed with the scheduled operation. Conversely, if the patient is old, obese, debilitated or has known cardiac disease, and if the agent was one which normally does not depress the heart, such as ether, circulatory collapse must be assumed to be ominous of serious impairment of the circulatory mechanism. The operation should be cancelled unless it is imperative for the patient's welfare. If it must be done, a minimal procedure should be selected; and, if possible, it should be postponed to a later date.

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REFERENCES

1. Adams, H. D., and Hand, L. V.: Twenty-minute cardiac arrest with complete recovery, J.A.M.A., 118:133-135, Jan. 10, 1942.

2. Bailey, Hamilton: Cardiac massage for impending death under anesthesia, Brit. Med. J., 2:84-85, July 19, 1941.

3. Barber, R. F., and Madden, J. L.: Resuscitation of the heart, Am. J. Surg., 44:151-168, May 1944.

4. Barber, R. R., and Madden, J. L.: Historical aspects of cardiac resuscitation, Am. J. Surg., 70:135-136, Oct. 1945.

5. Beck, C. S.: Resuscitation for cardiac standstill and ventricular fibrillation occurring during operation, Am. J. Surg., 54:273-279, Oct. 1941.

6. Eastman, N. J., Dunn, R. B., and Kreiselman, J.: Relative value of pure oxygen and of carbon dioxide mixtures in experimental resuscitation, Am. J. Obst. and Gyn., 36:571-579, Oct. 1938.

7. Prevost, J. L., and Battelli, F.: Sur quelques effets des discharges electriques sur le coeur des maniferes, Comp. rend. Acad. d. sc., 129:1267, 1899.

8. Thompson, S. A., Birnbaum, G. L., and Shiner, I. S.: Cardiac resuscitation, J.A.M.A., 119:1479-1485, Aug. 29, 1942.

9. Volpitto, P. P., Woodbury, R. A., and Abren, B. E.: Influence of different forms of mechanical artificial respiration on pulmonary and systemic blood pressure, J.A.M.A., 126:1066-1068, Dec. 23, 1944.

10. Weeks, C., Ryan, B. J., and Van Hoy, J. M.: Two deaths associated with supradiaphragmatic vagotomy, J.A.M.A., 132:988-990, Dec. 21, 1946.

11. Weinberger, L. M., Gibbon, M. H., and Gibbon, J. H., Jr.: Temporary arrest of the circulation to the central nervous system, Arch. Neurology and Psychiatry, 43:615-634, April 1940.